interval 1.02–2.57; p = 0.042) and the cut-off value determined from the receiver operating characteristic curve was −20.75% (area under curve, 0.928; 87.5% sensitivity, 87.5% specificity). Furthermore, changes in PSS among patients in whom these parameters were measured at least twice correlated significantly with changes in MPAP (r = 0.633, p = 0.037). Conclusions: PSS of the RV free wall might serve as a useful non-invasive indicator of PH.

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Prognosis of sleep-disorder breathing for chronic heart failure and the effectiveness of nocturnal home oxygen therapy and continuous positive airway pressure

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Background: Sleep-disordered breathing (SDB) has been reported to influence the mortality of patients with chronic heart failure (CHF). However the predictors of lethal cardiac events in CHF patients with SDB remain to be elucidated. Methods: We examined whether the severity of SDB was associated with lethal events in CHF patients, and whether the respiratory therapy for SDB improved the prognosis. Ninety-five patients with stable CHF who had the examination of SDB by overnight polysomnography between August 2000 and November 2008 were enrolled in the present study (62.3 ± 14.5 [SD] years-old, M/F 72/23). SDB events were quantified by the apnea hypopnea index (AHI). All patients with more than 10/h for AHI (n = 42) at the initial evaluation, were recommended the respiratory therapy (RT); oxygen therapy (HOT) or continuous positive airway pressure (CPAP). Endpoints were defined as lethal arrhythmic events (sudden death or ventricular tachyarrhythmia) or lethal events (overall death or ventricular tachyarrhythmia). Results: During 29 ± 17 months of follow-up, 18 patients died and 10 ventricular tachyarrhythmias occurred. The multivariate proportional hazard analysis showed that more than or equal to 5AHI was an effective risk factor for both lethal arrhythmic events (P = 0.026) and lethal events (P = 0.043). Second, RT improved significantly the number of AHI, but did not reduce both lethal arrhythmic and lethal event rates. However, 4 patients who reached less than 5 AHI by RT had neither lethal arrhythmic nor lethal events during follow-up period. Conclusion: SDB could be one of independent predictors of lethal arrhythmic events and lethal events in patients with CHF. Conventional RT might improve the prognosis of these events.

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Detection of hydroxyl radical in isolated Goto-Kakizaki (GK) rat arteries by trapping with 4-hydroxybenzoic acid

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Goto-Kakizaki (GK) rats were established as a model of inbred type 2 diabetes mellitus. We have shown that acetylcholine (ACh)-induced relaxation on aortic ring preparation and ACh-induced NO release from cultured endothelial cells in GK rats were reduced compared to Wistar rats. Furthermore, we have shown that norepinephrine (NE)-induced ATP release from cultured endothelial cells in GK rats were also reduced in GK rats. These differences may be related to vascular endothelial dysfunction in GK rats. Endothelial dysfunction and oxidative stress are the main pathophysiological mechanisms of diabetes mellitus complications, such as cardiovascular disease, renal failure and ischemia-reperfusion injury. In the present study, we investigated the generating of hydroxyl radical from Wistar and GK rat isolated aorta. 4-Hydroxybenzoic acid (4-HBA), a compound structurally very similar to salicylate, was introduced recently as a hydroxyl radical trapping agent. Hydroxylation of 4-HBA by hydroxyl radical yields a single product, 3,4-hydroxybenzoic acid (3,4-DHBA). 3,4-DHBA was quantified by HPLC-electrochemical detection technique. Spontaneous hydroxyl radical generation did not differ from Wistar and GK. NE and ATP significantly increased the hydroxyl radical generation from both arteries. There was no significant difference among the amounts of the hydroxyl radical generation, while NE and ATP did not alter the NO release. Hydroxyl radical generation did not correlate NO release. Moreover, the quantity of mRNA expression of NOS2 and SOD1 was almost the same. These results suggested that hydroxyl radical may be generated from vasculature (arterial smooth muscle and endothelium) and related to vascular dysfunction.

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